modifies a GM1 associated activity, wherein the agent is not coupled to an antigen, and the agent modulates at least one change selected from the group consisting of a change in antigen specific IgE levels, a change in antigen specific T-cell reactivity, a change in IgG levels, a change in IgA levels, and combinations thereof.

38. A method according to claim 37 wherein the agent binds to GM1.

39. A method according to claim 37 wherein the agent reduces levels of serum antigen-specific IgE.

REMARKS

Claims 1 to 19 were pending in this application in its published PCT format. The claims were cancelled and replaced by new claims 20 to 39 to put them into form for U.S. prosecution, replacing use claims with method claims, and to streamline the case and save considerable fees by re-writing multiple dependent claims as dependent claims. As amended, the application has a standard U.S. set of twenty claims, three of which are independent.

Newly presented independent claims 20, 32 and 37 particularly point out assay and treatment methods for identifying and using an agent useful in the treatment of an allergic or hypersensitivity condition comprising screening test agents not coupled with an antigen with a ganglioside receptor, determining whether the agent modulates gangliosideassociated activity, and identifying useful agents by observation of modulation of ganglioside associated activity. Support for the claims may be found in the specification on page 10 at lines 20 to 27, page 33 at lines 9 to 19, and page 34, lines 4 to 9.

Dependent claim 21 adds to claim 20, and claim 38, to 37, the limitation that the agent is a GM1 binding agent; support for the limitation can be found in the specification on page 12 at line 22. Claims 22 and 35 provide a list of agents set out in the specification on page 12 at lines 25 to 26. Claim 36 particularly points out a preferred embodiment wherein the agent is EtxB, as stated in the

specification on page 13 at line 2. Claim 23 points out another embodiment wherein the agents have an effect on GM1 mediated intracellular signalling events, but no GM1 binding activity, as described in the specification on page 29 at lines 23 to 25. Claim 35 combines the limitations of claims 21 and 23, as does language of the specification on page 29 at lines 21 to 25. Claims 24 and 33 point out agents that block an IgE mediated response, tracking the language of former claim 3. Claim 25 adds to claim 24, and claim 39, to 37, the limitation that antigen-specific IgE levels are suppressed; support for the limitation can be found in the specification on page 33 at lines 14 to 15. Claim 26 distinctly claims agents capable of enhancing the production of IgG and/or IgA, as described in the specification on page 34, lines 4 to 9. Claim 27 points out agents that reduce the production of Th2 associated cytokines as described in the specification on page 33 at lines 28 to 29. Claim 28 adds to claim 27 the limitation that the cytokine is IL-4 in some embodiments; support for the claim can be found in the specification on page 34 at line 1. Claims 28 and 29 point out agents that increase the expression of cytokines involved in down-regulating the allergic response, such as IL-10 or TGFβ as described in the specification on page 34 at lines 1 to 2. Claim 31 tracks the language of former claim 17. No new matter is presented.

The claimed invention provides significant new ways of treating allergic conditions and/or hypersensitivity conditions through the induction of specific immune deviation or suppression by the identification and use of agents exhibiting the properties set out in the specification. Applicants therefore request early and favorable consideration of the claims.

If the undersigned can advance the prosecution of this application in any way, the Examiner is invited to call at the number listed below.

Respectfully submitted,

MARY M. KRINSKY, Reg. No. 32423
79 Trumbull Street
New Haven, Connecticut 06511-3708
(203) 773-9544